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ORIGINAL ARTICLES.

DISEASE OF THE SMALL BLOODVESSELS AS STUDIED WITH THE OPHTHALMOSCOPE.*

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In the space available here it is only possible to call attention to the extent of this subject and to its importance. The vessels seen with the ophthalmoscope are all small vessels. The largest branches of the central retinal vein are rarely one-quarter of a millimeter (1-100 of an inch) in diameter. Seen under the magnifying power afforded by the cornea and crystalline lens, which varies from 12 to 20 diameters, the smallest arterial and venous branches, 0.01 mm. in diameter, (the size of a white blood corpuscle) become distinctly visible. In these vessels pathologic changes can be watched with the ophthalmoscope from day to day, or minute to minute during life. And, since many of the more common vascular diseases occurring in the retina lead to blindness with glaucomatous pain, these same vessels can subsequently be subjected to study by laboratory methods, of microscopy, sectioning, staining, chemical investigation, etc. It is therefore not strange that we have more exact and extensive knowledge of the effects of disease on the retinal vessels than of its effects on any other vessels in the body. Much of what is known of vascular disease as seen with the ophthalmoscope is of that general fundamental character that should be at the command of every student of medical science, every practitioner of the healing art.

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Vascular Spasm.—Cases occur in which the sight of one eye is suddenly lost for a few minutes, then vision gradually returns. The patient is usually alarmed by this occurrence, but before he can seek professional aid the return of vision has relieved his anxiety. A second attack naturally produces less alarm; and often several periods of temporary blindness have been experienced before the eyes are examined. When seen in the interval the eye presents no evidence of the condition. In a larger number of cases one or a few attacks occur; then they cease spontaneously and are forgotten, perhaps, without the case having even come under ophthalmoscopic examination.

In a few cases there comes an attack that is not followed by recovery. The blindness is permanent. Such a patient soon seeks expert advice, presenting an eye that shows the evidences of an obstruction of the retinal vessels. In a case of the kind which came to me the previous attacks had each lasted several minutes; and in this one it was not until his eye had remained blind for an hour or more that the patient became alarmed about it. I saw him the next day, when his eye presented all the evidences of complete obstruction of the central retinal artery. It remained entirely blind until his death a year later from typhoid fever.

Twenty years ago Benson[1] reported a case in which he had the opportunity of examining the eye with the ophthalmoscope during one of these attacks of temporary blindness, and found a large division of the retinal artery entirely bloodless for some distance from the optic disk. While he watched it the position of the bloodless portion shifted toward the periphery of the fundus, until it reached a large bifurcation, when it suddenly disappeared. On subsequent occasions with the same patient similar observations were made by others of the clinical staff of the hospital.

Ten years ago, while examining a patient who had complained of temporary obscuration of vision in the right eye, Jamieson[2] saw the retinal arteries fade into white lines, this being followed closely by fading of the veins, until he could not make out their position. After about two minutes the vessels again became visible, and returned to their normal condition. The patient stated that this was similar to the previous attacks.

The best case of the kind has been reported by Harbridge.[3] In his patient the attacks occurred for ten days, one day as often as every forty minutes. He was able to observe them

repeatedly himself, and to demonstrate them to Drs. de Schweinitz, Zentmayer, and others. There was gradual diminution in the calibre of the inferior temporal artery followed by the other arteries, until they were completely collapsed, and this was followed by a similar process in the veins. This condition continued about four minutes, then the arteries and veins filled in the order in which they had been emptied, and sight immediately returned.

In Lundie's case,[4] seen as the attack was going off, the reporter "could almost fancy that a tiny transparent finger and thumb were nipping the artery just as one nips an India-rubber tube." Hoppe[5] reported a case in which the lower temporal branch of the retinal artery appeared absolutely empty on the optic disc, and for one-quarter of the disc diameter beyond it, while peripheral to this the vessel was filled with venous blood. From time to time the emptied portion of the vessel varied in position, and the peripheral blood column was at times broken up. On the fifth day the blood column became bright red, and on the sixth day it had assumed its normal color, and the vessel was nearly its full width. Subsequently the previously empty portion of the vessel became over-distended, presenting a slight aneurysmal dilatation. Hoppe speaks of the condition as one of collapse of the vessel, but gives no satisfactory explanation of the mechanism of such collapse other than arterial spasm.

Most of the patients presenting this condition have been past middle age and have shown evidences of arteriosclerosis. My patient, however, was a man in good health and under 30 years of age. As to possible causes: Jamieson's patient, aged 84, had been taking oil of wintergreen and Haarlem oil, and had a pulse losing one to three beats per minute. Benson's patient was a man of 32, who had suffered from malarial and rheumatic fever. Lundie's patient was a man of 88, with rather feeble heart, but with arteries less hard than usual for his age. Harbridge's patient was a man of 49, who had used tobacco and alcohol to excess, and who subsequently showed evidence of arteriosclerosis. Hoppe's patient, a woman of 32, had been prostrated by a fright, and was delivered of a still-born child twenty-four hours later.

With reference to treatment: Most cases recovered from the single attack, and have subsequently become free from attacks without any clear evidence of benefit from the measures employed. Lundie's patient quickly recovered under the vasodila-

tor influence of a drink of whiskey. Harbridge's patient continued to have attacks regularly under nitroglycerine, potassium iodide and mercury. But they ceased absolutely and permanently after a thorough saline purge. It seems probable that some of the cases of so-called retinal embolism that have been cured by nitrites or massage of the eyeball were really cases of arterial spasm rather than of embolism.

The wider interest of these facts lies in their application to small arteries throughout the body. There is no reason to believe that arterial spasm is confined to retinal arteries. Indeed we know from Raynaud's disease that it is not. The phenomenon of migraine points strongly towards similar disturbance of circulation in the visual centers. The presumption is that spasm may occur in any small vessel in the body, the vasomotor function being liable to abnormality as any other. It is perfectly practical to ask, What symptoms are due to such spasm? Under what general conditions is it likely to arise? What are the general causes, and how are they to be removed? The ophthalmoscope has made its contributions to this branch of pathology. A better understanding of the retinal changes may have to wait until similar phenomena have been studied in other parts of the body

Permanent Obstruction of the Retinal Circulation.—This was first described by Graefe in 1859. Graefe's patient suffered from endocarditis with aortic obstruction; and the diagnosis of embolism of the central retinal artery seemed fully justified. But the ophthalmoscopic symptoms that followed, clouding of the retina about the posterior pole of the eye, the cherry-red spot in the macula with partial collapse of the veins, are symptoms that we now know also arise from obstruction of the retinal circulation due to causes other than embolism. A great many cases have been reported under the heading embolism of the central retinal artery that were certainly not of that nature, but were due to these other causes. The diagnosis of embolism must rest indeed rather on collateral evidence than upon the changes visible with the ophthalmoscope.

A certain small minority of these cases doubtless are due to embolism. But the majority probably arise from thrombosis of the artery starting in points of endarteritis, while closure of the artery is also likely to occur after thrombosis of the central retinal vein. Harms has pointed out that obstruction of both arteries and veins is frequently the condition found in the eyes

that come to removal for absolute glaucoma. The precedence of preponderating arterial or venous obstruction is indicated ophthalmoscopically by the absence or presence of retinal hemorrhages.

Complete obstruction of the central artery is followed in a few seconds by blindness which generally is permanent. The arterial branches show little immediate change. They do not collapse and the blood column is but little altered in color. On the other hand the veins become greatly contracted or flattened in some parts, while other parts may still be partially distended by blood that can be seen to move toward the optic disc.

The other striking early ophthalmoscopic appearance of arterial obstruction is opacity of the retina, which makes the margin of the optic disc hazy, and gives the general fundus near the posterior pole a pale or whitish appearance, interrupted by the cherry-red spot at the fovea and gradually fading out toward the periphery of the fundus. This opacity has generally been spoken of as œdema of the retina. But there is little positive evidence of swelling of the retina.

Hancock[6] suggested that the appearances were better explained by the occurrence of a tissue necrosis affecting the superficial layers of the retina. In a case reported by Shoemaker,[7] which terminated in death eighteen days after obstruction, Hosmer found the degeneration amounted "to ischemic necrosis." The same view has been endorsed by Coats,[8] and seems to me much more rational than the theory of œdema. The necrosis is confined to the superficial layers of the retina, the nerve fibres and ganglion cells. Where these are absent, as in the fovea, no change in the color of the fundus takes places. It is admitted that the very similar appearance seen in amaurotic family idiocy is due to degenerative changes in these layers. And similar veiling of the fundus without œdema has attended the toxic alterations in experimental poisoning with methyl alcohol.

Primary thrombosis of the retinal vein, a condition which does cause retinal œdema along with multiple hemorrhages, does not cause the peculiar veiling of the central part of the eyeground as we see it in arterial obstruction. The later atrophic changes that follow obstruction of the retinal vessels may be quite the same whether the obstruction has been previously arterial or venous.

Partial obstruction of the retinal circulation is more common

than is usually recognized, for the ophthalmoscopic symptoms characteristic of complete obstruction are lacking. In the arteries there may be points of narrowing from endarteritis. The obstructed veins may show a general distension with little or no hæmorrhage. In neither case is there complete blindness, and the prognosis is good.

One of my patients, a woman of 42 with marked evidence of endarteritis, had been given a prognosis of permanent blindness following an attack that may have been partly due to arterial spasm, or may have followed a hæmorrhage into the optic nerve sheath. Although her vision was greatly reduced at first, it subsequently rose to 4/4 mostly, in a small field, and she retained a useful eye until the time of her death four years later.

A man of 42 with vision reduced to 1/10 of normal, complained of shifting interruptions in his field of vision. He saw in certain places "as through rifts in a cloud." There was an uncertain history of early syphilis. The retinal veins were large, tortuous, and markedly irregular in calibre. The retina was distinctly hazy in patches, but not universally so. The arteries were relatively narrow. Amyl nitrite brought up vision to 4/25. After about three weeks vision rapidly improved to normal, and it remained normal while he continued under my observation, a period of twelve years.

Angiosclerosis.—In a large proportion of cases obstruction of the retinal circulation is associated with changes in the vessel walls. The fundus appearances of arteriosclerosis and of the associated condition of renal retinitis have been so frequently described that they need not be dwelt upon here. But a few points that ought to be of interest to the whole profession may be referred to. As Coats observes, the arteries show endarteritis producing irregular narrowing of the calibre of the vessel. The veins show rather a periphlebitis, hiding the whole blood column by a more or less opaque coat.

As was pointed out by Bull[9] disease of the vessel walls is more likely to cause hæmorrhage at an early stage than later. This observation has been repeatedly confirmed in my experience. The general explanation has been that the later thickening of the vessel walls enables them to withstand the pressure, preventing hæmorrhage. Coats suggests that the activity of the arterial circulation is diminished in the later stages of arteriosclerosis. With reference to these ophthalmoscopic evidences of angiosclerosis the profession at large needs to ponder these re-

marks of Werner:[10] "Every general practitioner admits the necessity of ophthalmoscopic examination in cases, say, of suspected intracranial tumor, whereas, I take it, comparatively few are aware of the importance of such an examination in the class of cases now under discussion. But yet, if we stop to consider for a moment, a cerebral tumor is, comparatively speaking, a rare affection and its treatment unsatisfactory, whereas angiosclerosis is a very wide-spread disease and one in which medical advice and treatment, especially in the incipient stages, may frequently be the means of prolonging life for many years."

Aneurysm.—The early work on arteriosclerosis in the large vessels was so closely associated clinically with aneurysm that most physicians expect to find aneurysm as one of the common evidences of angiosclerosis of the retina, but in reality it is rare. Gowers[11] reported a case and referred to others previously reported. Since then cases have been reported by Fisher.[12] Hoppe's case[5] of secondary dilatation of the vessel has been mentioned above. Leber[13] divides miliary aneurysm into two groups: those alluded to above, associated with senile changes; and another group in which multiple aneurysms have been associated with retinal changes resembling circinate retinitis.

Angiomatosis.—A distinct and extremely interesting type of vascular disease has gradually emerged in the literature. It has been known widely in Germany as Von Hippel's disease of the retina, but is called by him *angiomatosis.* Cases had previously been reported by Fuchs, Wood, Collins, and Darier. But it was scarcely recognized as a definite entity until Von Hippel[14] described his case and exhibited it before the Heidelberg Ophthalmological Congress; and later, after watching its progress for sixteen years, was able to examine the eyeball microscopically. The ophthalmoscopic appearances include a large, rounded, reddish body in the retina, entered by one or more dilated tortuous arteries and their accompanying veins. One part after another of the retina becomes affected. The retina becomes thickened, altered, and detached. All sight is lost and ultimately glaucoma intervenes, so that the eyeball has to be enucleated. Seven such eyes have now been studied microscopically.

Recently Meller[15] has put forth the view that the condition is not primarily a vascular disease; but is essentially a disease of the glial tissue of the retina, accompanied by vascular changes. Perhaps only extended collateral studies can decide the question

of its essential nature, but it illustrates the extremely close association of diseases of the various parts of the body with vascular conditions. The recognition of angiomatosis as a distinct disease was hindered by its inclusion in the large group of cases of retinitis with massive exudation, some of which are closely associated with disease of the vessels. Retinitis with massive exudate is probably due to various causes. But some cases have been traced apparently to tuberculosis involving especially the vessels.

Tuberculosis of the Retinal Vessels.—Both massive exudation and recurring retinal hæmorrhage in young persons have been more and more traced to vascular disease, and particularly to tuberculosis. Cords[16] reports a case of retinal periphlebitis which rapidly recovered under tuberculin treatment, and brings together six others probably tuberculous in character. His case showed periphlebitis, the blood column being in certain parts hidden by a white sheath; and the veins irregular in calibre with peculiar tortuosities, tufts of newformed vessels, anastomoses, and hæmorrhages.

Arnold Knapp[17] records four cases regarded as tuberculous, of which one recovered and another improved under tuberculin treatment. These latter were characterized by recurring hæmorrhages into the vitreous. Fleischer[18] records the case of a man suffering from pulmonary tuberculosis, in whom the lesions described by Cords were present in the arteries of both eyes. One eye developed tuberculous lesions in the anterior segment, became glaucomatous and was removed. The microscope showed that the retinal lesions were also tuberculous, some of the veins being surrounded by epithelioid and giant cells.

Syphilis of Retinal Vessels.—Retinal vascular disease due to syphilis is probably more common than that due to tuberculosis. But it has been more frequently described, and therefore needs but little space here. Usually it is recognized under the heading of retinitis; or of chorioretinitis, since the superficial vascular layer of the choroid is very likely to be involved. The circulation through the smaller choroidal vessels becomes interrupted, the vessels largely disappear, and the external layers of the retina atrophy; while the pigment cells disappear, or migrate, forming masses that may be located even on the inner surface of the retina. These changes are seen in both congenital and acquired syphilis.

Acute Infections.—Many acute infections, notably influenza, puerperal septicæmia, scarlet fever, and cerebrospinal meningitis, are liable to cause metastatic ophthalmia and panophthalmitis. The majority of such cases are seen only at a late stage, the general gravity of the disease and the apathy of the patient causing the earlier stages to be overlooked and neglected. But in rare cases like those reported by Rössler[19] and Wiegmann[20] the ocular lesions have come under observation early, and these cases indicate clearly their vascular origin and the general importance of the blood vessels in metatasis. In the retina points of vascular obstruction appear, probably starting with minute bacterial emboli, and quickly extending by thrombosis. These points become foci of local infection, quickly involving all the ocular tissues in the process, which may or may not be suppurative.

Hæmorrhage.—Regarding hæmorrhage from minute vessels the ophthalmoscope has revealed many things that are of general significance and importance. In the retina hæmorrhage is practically a constant phenomenon of acute and subacute inflammation. No sharp line separates transudation of the formed elements of the blood from that of the fluids. The retinal circulation is a terminal one. From the freely inosculating vessels of the choroid hæmorrhage is less common, although it probably occurs more frequently than is recognized. The regressive changes undergone by effused blood we have all watched with the ophthalmoscope. Here, too, the similarity of behavior to that of other exudates into the tissues is striking. The clot becomes decolorized and cannot be distinguished from a non-hæmorrhagic exudate; and its ultimate fate may be the same complete removal or retention as a white mass that has undergone fatty change.

Ophthalmoscopic examination has proved that hæmorrhage into the retina at birth is quite common. Coburn[21] examined the eyes of 37 infants still-born or dying before the age of 22 days, and found retinal hæmorrhages in 17. He collected from various sources the results of such examination in about 700 cases and found hæmorrhage in 20 per cent. Stumpf and Von Sicherer[22] examined the eyes of 200 new-born children, finding hæmorrhage in the eyes of forty-two. Closely allied to the pressure occurring during birth, is the pressure sustained in certain accidents, as where the body is buried under a heavy weight of earth, causing intense venous congestion of the head

and neck as reported by Béal.[23] More obscure are the causes of hæmorrhage into the retina seen after fracture of the skull, without direct injury to the eyes in the series of cases reported by Fleming.[24] The part played by hæmorrhage in causing retinitis proliferans is interesting and suggestive. But if, as is now urged, the vitreous is not of mesoblastic origin, but an outgrowth of the glial tissue of the retina, what happens in this condition may not throw much light on the sequels of hæmorrhage in other parts of the body.

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A NEW CONJUNCTIVAL FLAP INCISION IN CATARACT EXTRACTION.*

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In operations that require perforation of the globe, and in accidental perforating injuries, the present day procedure with the majority of conservative ophthalmic practitioners is to cover the corneal or the scleral wound with some form of conjunctival flap, the object being to prevent infection and to secure a rich blood supply in order to obtain rapid union of the divided surfaces. In the extraction of senile cataract the ordinary method of protecting the marginal wound that is made in the cornea, when any of the linear knives are employed, is to continue the incision in the conjunctiva for several millimeters from the apex of the corneal incision. More elaborate methods of obtaining the conjunctiva flap are those of Czermak, Dimmer, Bajardi, Cluckie and Van Lint; but they are also more difficult of execution. The one here to be described is as simple to obtain as that ordinarily employed. However, in order to produce it, the double fixation forceps, an excellent instrument not in common use, is necessary.

With this forceps the conjunctiva, over spaces 5 to 7 millimeters in width to either side of the cornea, is grasped in such a way as to cause the loose conjunctiva to fold moderately over the corneal margin. If a Beer or a Graefe knife is employed, or any of the long narrow knives, the corneal incision is made as usual, just back of the clear cornea in the sclero-corneal margin (Fig. 1, A). This will naturally involve the folded conjunctiva. After leaving the apex of the corneal wound, the knife may be continued in the conjunctiva as far as may be desired by the operator. Personally, I never care to have this apical flap longer than 1.5 to 3 mm., as it is liable to become entangled with the iris in making the iridectomy, and also from a more extensive surface there may be hæmorrhage that will obscure the lens at the time of the division or the removal of the anterior capsule. When the incision has been completed and the forceps are removed, the course of the corneal wound will lie in the sclero-corneal margin, as indicated by the dotted line, and that of the conjunctival wound will be well in the conjunctiva as indicated by the irregular continuous line in Fig. 1, B. This provides a

*Read before the Washington University Medical Society, April 12th, 1915.

conjunctival covering to the corneal wound at the entrance of the knife, and at the exit of the counter puncture, which cannot be obtained in the ordinary conjunctival flap method, except by placing the corneal incision dangerously near the ciliary border; while in the nasal and the temporal quadrants of the wound, if the apex be vertical, the flap spreads out from the cornea like small wings, as a corneal protection.

The employment of the lance-shaped keratome renders the procedure practically subconjunctival, as there is no division of the conjunctiva at the nasal and temporal angles. Also the shortest portion of the flap is at the center, where it is most to be desired to facilitate the iridectomy, the removal of the capsule and the expulsion of the lens. The entrance point of the knife

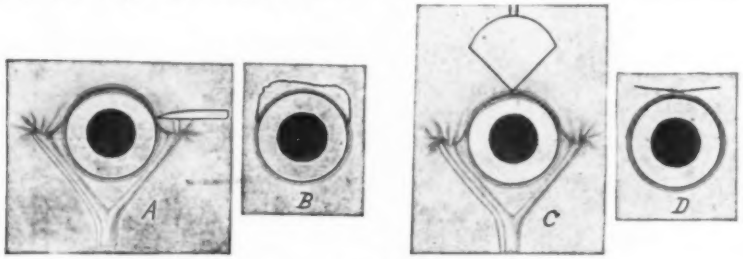

Fig. 1.

A new conjunctival flap incision in cataract extraction. In the diagrams the shaded ring illustrates the actual antero-posterior distance from the clear corneal margin to the canal of Schlemm. The dotted line represents the course of the incision in the cornea; the continuous line the incision in the conjunctiva.

A.—Illustrates the folding of the conjunctiva over the cornea by means of the double fixation forceps, and the puncture with the linear knife.

B.—Illustrates the completed incision with the linear knife. The conjunctival incision may be extended upward if desirable.

C.—Illustrates the folding of the conjunctiva over the cornea and the puncture with the lance knife.

D.—Illustrates the completed incision with the lance knife.

and the completed incision are illustrated in Fig. 1, C and D. These wounds frequently heal so kindly that the conjunctiva on the opposite side of the cornea scarcely changes color during the healing interval.

In making the incision with any form of knife, it should always be kept in mind that the incision is to be placed just back of the union of the conjunctiva with the cornea, and as far forward from the iris-cornea angle and the canal of Schlemm as possible, so as to have the least interference with the intraocular circulation following the operation. The relative actual vertical depth of this space is about 0.75 mm., and it is represented by the shaded line surrounding the clear corneal margin in the accompanying diagrams.

ON THE MYOTONIC CONVERGENCE REACTION OF THE PUPIL.*

By Professor Dr. H. Oloff.

The myotonic convergence reaction is rare among the abnormal forms of pupil reactions, and little attention is being paid to it even in ophthalmological circles.

As is known, this reaction is characterized by the fact that after convergence it takes a longer time than in the norm for the pupil contracted by this convergence to dilate when looking at the distance.

This phænomenon has ætiologically nothing in common with myotonia congenita (Thomson's disease). Only in one exceptional has Hoche observed a similar reaction in true myotonia. Proposed by Saenger, the name of myotonic reaction was accepted only because after contraction the sphincter pupillæ remains similarly in a prolonged state of contraction as the voluntary muscles do in Thomson's disease.

The myotonic character of the convergence reaction is often overlooked and the wrong diagnosis of loss of light reflex is made. Against this error the fact alone speaks, that with loss of the light reflex the convergence reaction is usually very prompt, because it is even physiologically stronger than the light reaction.

The opinions still differ greatly concerning the details of the myotonic convergence reaction and its relations to other similar pupillary phænomena. This and the rare occasions at which the myotonic convergence reaction is observed prompt me to consider it in the following more in detail, in describing two cases which I have lately had occasion to examine and observe more closely.

I have to thank Professor Dr. Siemerling, Director of the University Neurological Clinic at Kiel, for the second case.

Case I.—P. R., 18 years old, entered the neurological department of the Marine Hospital at Kiel on January 7th, 1914. He was referred to the chief physician, Dr. H. Schmidt, to be examined as to his mental condition since he had of late done his service in an unsatisfactory manner. His mental faculties were found to be slightly below par and, according to his brother's statement, this had been noticed since his childhood. He had

*Klin. Mtsbl. f. Augenhlk., October, 1914.

never been seriously ill, never had received an injury to his head. As the brother further stated, his pupils have been unequal since childhood.

On examination of the eyes I found: Palpebral fissures of equal width, motility of eyeballs free. Well marked inequality of the pupils; the right one 5 mm., the left 7.5 mm. wide. Pupils not round. Direct and consensual light reaction positive, L.E. very sluggish.

The convergence reaction of both pupils positive, yet it is noticed that the convergence reaction in the left eye is slow and tardy and that after the change from the convergence position the left pupil remains contracted for one minute longer, while the right pupil at once dilates as in the norm.

Visual acuity, accommodation, fundus, etc., are normal for both eyes. The iris is colored normally and shows no signs of atrophy or previous inflammation.

During the further course of the examination the width of the pupil is strangely changed so that the right pupil is suddenly dilated and reacts more slowly to light than the left one. After the now following convergence reaction the right pupil remains contracted for a longer period after the change from the convergence position, just as the left pupil had done before.

This strange change in the width of the pupils and their reactions repeated itself apparently absolutely without rule. Now the right pupil, now the left one is the larger, and every time the larger pupil shows the symptoms of sluggish, sometimes altogether absent reaction to light and of a marked myotonic convergence reaction.

An examination of the remainder of the nervous apparatus only shows the patellary reflexes wanting. Nothing abnormal in other organs.

Wassermann of blood and cerebrospinal fluid is negative. The cerebrospinal fluid is perfectly normal.

Case II.—R. B., 44 years old, fish dealer at Kiel, was treated at the Kiel University nerve clinic for chronic alkoholism from March 10th to April 2nd, 1914. Had been previous to this four times at this clinic for the same and on account of an injury to the head on May 5th, 1905.

Stat. praes. On the left side of the head where the occipital and parietal bones meet a narrow, bent, moveable scar which is still sensitive to pressure.

There is no difference in the size of the palpebral fissures.

In the extreme lateral positions, especially to the right, moderate nystagmus. Otherwise movements free.

Both pupils not quite round. Right pupil 5.5 mm., left 3 mm. Light reaction, direct and consensual, positive, but not very great; left eye direct and consensual almost absent.

In the near vision the left pupil contracts slow, but in the end more than the right one. When looking again at the distance the left pupil for some time (about 30 seconds) remains contracted and then dilates slowly, while the dilatation of the right pupil after the cessation of convergence dilates promptly (myotonic convergence reaction of the left pupil).

Function and accommodation of both eyes good. Left disc temporarily slightly pale. Everything else normal.

The only other deviation from the norm is that the knee reflexes which were still present in 1910, that is 5 years ago after the injury, can no longer be evoked. No other signs of tabes, paralysis or any other spinal or brain affection.

As far as the eye-symptoms are concerned these two cases have in common (1) difference in the width of the pupils, and (2) monolateral myotonic convergence reaction, the light reaction on that side being absent or almost absent. While, however, in the one case (R. B.) the myotonic convergence reaction was always found in the smaller pupil of the left eye, in the other case (P. R.) this reaction was seen at times in the right at others in the left eye, every time after, immediately previous to it, this respective pupil had suddenly dilated and then on light stimulus had become nearly or totally without reaction.

A similar diminution of accommodation, which on acount of the close relation between convergence and accommodation had to be thought of, could not be demonstrated in either patient.

There were no visible local alterations in the iris (atrophy, posterior synechiæ) which, as sequelæ of a former iritis, might have been accused of producing the myotonic convergence reaction.

On the contrary in both cases the accompanying symptoms—difference in width of pupils, diminution of light reaction, lack of patellary reflexes, further in the one case lowered mentality, in the other a previous head trauma—point with the greatest probability to a cerebral cause. There were, however, no distinct localizing signs of an organic focal affection, as all other symptoms were wanting.

Roenne in 1909 has critically collected 11 cases of myotonic

convergence reaction of the pupils, observed and described by him, Axenfeld, Saenger, Strassburger, Nonne, Rothmann, Roemheld and Marcus. Dimmer reported a similar case in 1911 and Lerperger one to the Vienna Ophthalmological Society on May 11th, 1914.

Furthermore cases of Piltz, Strassburger, Saenger and Bach are shortly mentioned in literature.

From these cases it is seen that the picture of myotonic convergence reaction is by no means a uniform one.

Two chief forms have to be differentiated clinically. In the vast majority of the reported cases the picture is confirmed to a slower disappearance of the convergence reaction and was simply a pupillary symptom. The accommodation was normal. The slow convergence reaction may be due to the fact that both phases of the reaction, contraction and disappearance of it take an abnormally long time or that at least one of these two phases is normal. From present observations the former seems to be the rule. Only in one of the cases collected by Roenne the contraction phase was mentioned as having been normal.

The second chief form of myotonic convergence reaction is combined with a more or less marked paresis of accommodation on that side. This has been seen in 3 cases (Roenne, Dimmer, Lerperger). In Dimmer's case the pupil dilated at once normally after the stopping of the convergence reaction, but the beginning of the reaction and the accommodation were abnormally slow. In the cases of Markus and Strassburger it is stated that only the relaxation of the accommodation was slow. In Nonne's case the action of the accommodation is uncertain.

In three of the 15 reported cases both eyes showed myotonic convergence reaction while the accommodation was normal. As is seen from Roenne's cases the myotonic convergence reaction was observed in both eyes in two cases, while in one of my cases (R.) a continued change of the width of the pupil was noticed so that always the wider pupil alone reacted myotonically on convergence. I could find no other such case in literature. In the remaining 12 cases the other eye was normal as to the pupil reactions, in the twelfth case there was lack of light reaction.

Some of the cases collected showed a surprising uniformity between the orbicularis reaction and the myotonic convergence reaction in that the pupil when contracted through energetic closure of the lids also enlarged again, but very slowly.

The appearance of the myotonic convergence reaction is espe-

cially characteristic when it concerns only one eye. In consequence of the slower contraction the affected pupil may sometimes in these cases reach a higher degree of miosis than the healthy pupil in the other eye.

The light reaction was uniformly very much reduced in the eye reacting myotonically on convergence or altogether wanting. This condition has by some experienced observers (Bach, Bumke) been mentioned as characteristic for the myotonic convergence reaction.

From this and from the fact that the accommodation is, also, affected Roenne concludes the hitherto observed cases of myotonic convergence reaction belong to a common series which in reality are continuous changes from the loss of light reflex on one to ophthalmoplegia interna on the other side. Bumke does not agree with this view because the very presence of a myotonic reaction in itself excludes a loss of the light reflex. He looks upon the myotonic convergence reaction as a "not very frequent modification of an absolute loss of light reflex" because the myotonic convergence reaction is more powerful than the light reflex action and because "in general nerve pathology elective paralyses of certain muscles are generally known." Even cases of oculomotor and sphincter paralysis in which the pupil lacking the light reflex contracts somewhat on a large convergence innervation, cannot be adduced in favor of Roenne's opinion. They do not belong here, as Axenfeld insists, and they are explained by the fact that the convergence reaction forms a stronger stimulus.

A similar slowing up of the reaction as in convergence is in rare cases seen also when illuminating the pupil. It is understood that in such cases the pupil must still react to light. Since this is not or very incompletely the case in myotonic convergence reaction, as we have seen, it is at once apparent that both forms of reaction are impossible in one and the same eye.

Pilz was the first to see such a tonic light reaction of the pupil, in 1900, in a paralytic and described the case. It was the left eye. While the pupil contracted fairly well to light, the subsequent dilatation began very slowly and gradually. The convergence reaction was prompt every time.

He, furthermore, saw other cases in which with loss of light reaction the reaction of the pupil in convergence, accommodation and contraction of the orbicularis was extremely slow. He finally succeeded in changing an actual pupil difference experimentally in such a way that at times the pupils became equally wide

In tonic light reaction, too, according to present observations, the picture is not altogether uniform. According to Bach this peculiar phænomenon of slow light reaction was seen mostly in cases of growing amaurotic loss of light reflex, more rarely in reflectory loss of light reflex.

Bumke and Bach observed this tonic light reaction a number of times in normal eyes with seemingly perfectly normal general nervous apparatus.

With this neurotonic reaction an anomally recently described by Weiler must not be confounded, in which the light reaction in paralytics, as far as it still exists, sometimes in spite of continued illumination persists but for a short time, and gives very rapidly place to dilatation. Bumke sees in this anomaly a precursor of loss of refractory light reaction.

We must certainly sharply differentiate between the neurotonic pupil reaction and the myotonic convergence reaction. This should be made apparent by naming them differently. Bumke in this anomaly in light reaction puts the main emphasis "not so much on the slow course," as "on the persistence of the pupil contraction." In an analogous manner he called the unusually long pupil contraction in the convergence reaction in pupils without light reflex the "myotonic convergence reaction," while Roenne and Dimmer speak in quite a general way of the "tonic reaction of pupils without light reflex" and include such cases which are characterized simply by a slowly beginning pupil contraction. According to Dimmer in cases in which the accommodation, too, is affected, we would have to speak of a "tonic reaction of the ciliary muscle."

As cause for the development of the myotonic convergence reaction Raeke in his "Diagnostic" mentions a trauma to the head. Of the 15 cases on which this paper is based two only (Axenfeld and Oloff) were of a traumatic nature. That traumata to the eyeball, optic nerve, brain and spine can cause paralysis of the pupil has been known for a long time. Among 133 cases of loss of light reflex Uhthoff found three after injury to the head or to the spine; it is, however, not plain from his account whether the loss of light reflex was amaurotic or reflectory.

Axenfeld was the first to note the occurrence of true, post-traumatic loss of light reflex. In the few cases observed by him this was always one-sided; the direct light reaction was wanting, the consensual and convergence reaction were normal. The motor innervation of the injured eye and the optic reflex arc

from the other eye were therefore preserved. He concludes rightly from this that there are separate pupil fibres present in both optic nerves, agreeing with Bach and Schirmer, who had made analogous observations after inflammations of the optic nerve. From this it is possible for a reflectory loss of pupil reaction to appear as well after a trauma as after an inflammation of the optic nerve, because the pupil fibers in the optic nerve, that is their terminal expansions, have by one of these causes lost their function.

One of Axenfeld's cases of traumatic loss of refractory light reaction, which is also described by Roenne, showed in the same eye a marked myotonic type of convergence reaction. Since, however, the trauma had also produced a slight atrophy of the iris tissue Axenfeld is inclined to assume for this special case rather a local damage to the iris than the isolated lesion of the pupil fibres in the optic nerve.

There are cases, according to Axenfeld, in which a lesion of the oculomotor destroys the direct and consensual light reaction, while a prolonged convergence innervation still produces a gradual, if limited, mydriatic pupil. The dilatation coming on when again looking at the distance may be so slow that the whole reaction takes on a myotonic aspect. For this it is not sufficient for the patient to converge only for a moment, he must look at an object for a prolonged time at the usual reading distance. As Axenfeld states, this picture of a myotonic type of convergence reaction usually lasts longer than all other paralyses of eye muscles.

In the case of B., which I described above, we probably can in the first place assume an organic change in the brain due to the severe head trauma to have been the cause of the myotonic convergence reaction. The large scar due to the falling of a beam upon his head, is even now—9 years after the injury—very tender to pressure. He is, however, also an alkoholic and at the last examination the knee phænomena were absent, yet there was so far no other sign of tabes or paralysis.

It should be mentioned that in two other of the reported cases the lack of patellar reflex has been mentioned. One, R., above described by myself, showed symptoms of lowered mentality observed since childhood. A lumbar puncture and the examination of the general nervous apparatus did not show any organic changes in the brain or spine.

In two cases no cause is mentioned.

In the last nine cases of myotonic convergence reaction the following certain or probable causes were noted, one each of tabes (tabes? paralysis?), multiple sclerosis, diabetes (tabes?), migraine and attacks of fainting, neurasthenia, morbus Basedowii, alkoholism, morbilli.

Aside from these there are several short communications in literature by Strassburg, Saenger and Hoche. Strassburger saw monolateral myotonic convergence reaction in two children with congenital lues, Saenger "in several cases of tabes and paralysis." Hoche observed this reaction in a case of true Thomson's disease.

From all this it is clear that aside from a trauma several other causes are thus far not well recognized, may be active in producing the myotonic convergence reaction. That absolutely normal eyes may show myotonic convergence reaction as Bumke and Bach state it to be the case with neurotonic reaction, may from present observation probably be denied. In the vast majority of cases we must, it seems, assume a central origin. This is supported by the fact that in all of the cases the light reaction was nearly or totally wanting. In one of my cases there was the unique condition that sometimes one and then again the other eye—seemingly without any rule—showed the symptoms of myotonic convergence reaction after the pupil had previously suddenly dilated and become immovable.

According to the observations of Axenfeld and Bach we may repudiate the possibility of an isolated affection of the pupillary fibers in the optic nerve in those cases only in which a trauma influencing the optic nerve or an optic neuritis has preceded.

Dimmer assumes different affections of the nuclei of the oculomotor nerves or of the centrifugal parts of the reflex arc coming from them.

According to whether the affection attacks preferably the nucleus or fibers of the oculomotor which transmit the light reflex, or the convergence contraction or the accommodation, the result will be a neurotonic light reaction, or a myotonic convergence reaction, or a tonic ciliary muscle reaction.

Rothmann sees the origin of the myotonic convergence reaction in a gradually developing atrophy and contraction of the paretic sphincter iridis which, like Dimmer, he thinks is due to an affection of the several parts of the oculomotor, while Saenger believes in a more peripheral cause.

As far as can be stated from present observations the myo-

tonic convergence reaction of the pupil must always be looked upon as a pathological phænomenon, yet till now it possesses no diagnostic value, neither as to the seat of the affection nor clinically.

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ON A PRIMITIVE TELESCOPE AND ITS USE FOR AMBLYOPIC PATIENTS.*

By Dr. W. Reitsch.

The use of two lenses as a primitive telescope is not new. Here and there at fairs such instruments are sold which, however, on account of their clumsy form and their low magnifying power, seem not much more than playthings.

Yet, the combination of a convex and a concave glass for magnifying purposes, according to the principle of the Hollandish telescope, is of decidedly practical value. It is only necessary to select a combination which will give a considerable magnifying power and the glasses must be mounted so that they can easily be carried about the person. This is of special advantage compared with a stable telescope with which, on the other hand, the primitive telescope as regards the quality of the image cannot compare. It would, however, be quite erroneous to assume that with skillful handling of the primitive telescope this difference in quality is of practical importance. Certainly the visual field is smaller than with a primitive glass; a slight chromatic and spheric aberration is noticed now and then, yet with skillful handling never to such a degree that the enlarged object does not appear in useful clearness.

In order to explain the usefulness of such a simple system, I wish to say that with such a primitive telescope with a magnifying power of 8 times small print—Nieden 5, even 4—can be read at a distance of 6 meters.

No very great skill even is necessary. I have often taken occasion to convince myself of this. Of course, there are clumsy people with stiff wrists to whom the skillful handling of the glasses is quite difficult. I may say even, that there are some who can never learn it. It also is more difficult for a trembling hand. The majority, however, has no difficulty in applying such a telescope at once and others just need a little exercise. For some it will mean an art which has to be practiced, but it is well worth to be learned.

According to my experience the most favorable combination is that of a concave glass of —18 D. with two convex glasses of 3 D. and 2.25 D. With this three different magnifying powers are obtained. For the distance a magnification of 6 times is usually sufficient, which is obtained with +3 D. With +2.25 D.

*Klin. Mtsbl. f. Augenhlk., September, 1914.

objects are magnified 8 times, but the visual field is correspondingly smaller. For near objects both convex lenses are used together and with them we have then +5.25 D. and magnification of three times. With this last combination we obtain easily what with a good telescope is possible only by an additional lens.

The handling of this primitive telescope is very simple. When using it attention must be paid to it that the lenses are held vertically to the visual line and as centrically as possible. The ocular (the concave glass) is held by the right hand when the right eye is used, by the left when using the left eye. This ocular is first held before the eye, then under guidance of the unarmed eye the convex lens is brought into the direction of the object, and by slow forward and backward movements the correct distance is found. It is best during this manœuver to close the unarmed eye and use it only by squinting for the control of the direction.

Since one eye can be used for guiding it is with a little exercise easier to catch the object with this primitive telescope than with a binocular one. If at first the image is indistinct, although the distance is correct, this is due to an oblique position of the lenses. It is then best to at first correct the position of the concave glass. If this does not make the image sharp a slight turning of the convex lens in different planes is easily successful. Under certain conditions it may be best to remove the objective lens (the convex) altogether, and then again put it in the position vertically to the visual line.

The possibilities for using the primitive telescope are the same in a general way as those of every other telescope. But it is a means with which we can assist amblyopic patients, and especially poor amblyopic patients.

The telescopic spectacles of Zeiss with a magnifying power of 1, 3 to 2 times are undoubtedly very useful, but can be ordered only in extremely rare cases. Their high price usually forbids their use. When this is not the case the patient objects to their appearance. The small magnifying power is usually not considered of sufficient value to offset the small visual field and to a certain degree, also, the unsightliness.

A good telescope, however, which would magnify sufficiently cannot be always carried on the person and in by far the majority of the cases is too costly. This gap can be very well filled by the primitive telescope. It gives an enlargement of from 6 to 8 times, can, like the watch, always be carried along and is so

cheap that it may occasionally be ordered as spectacles for a poor patient.

Of course, the amblyopic can only be made to see clear for a time. He is, however, grateful for such moments of clear vision and can find his way alone under circumstances under which he would otherwise be dependent on others.

I have at present under my treatment a farmer, 56 years old, who has lost his better eye by detachment of the retina and whose other aphakic eye with correction has only 1/15 vision with a fairly good visual field. He is not a skillful man, but has learned in a short time to use this telescope which gives him more than 1/3 vision, so that he is able to recognize signs in town and on the roads, and people.

I have had this primitive telescope made in two combinations. For ordinary purposes the combination of a concave lens with one convex lens (—18 D.⊃+3 D, magnifying 6 times) will probably be sufficient and be the best also, on account of the cheaper price. In other cases from the reasons stated above (3 different magnifying powers and the possibility of seeing near objects) the combination lens is to be recommended.

Nitsche and Guenther of Rathenow manufacture this pocket telescope.

A CASE OF PARESIS OF THE ACCOMMODATION AFTER DIPHTHERITIC VULVOVAGINITIS.*

By Dr. R. Pape.

On March 7th, 1914, a twelve year old girl was brought to me because for two weeks she had not been able to see well, especially reading and writing were impossible.

An examination showed R.E. V.=6/15, with +1 D.=5/5; L.E. 5/15 partly, with +1.5 D.=5/5.

Medium large print (Nieden 7) could even at an increased distance not be read without glasses. With +3 D. R.E. and +3.5 D. L.E. Nieden 1 was read easily at 30 cm. The pupils reacted well.

With this condition the first thought was of a post-diphtheritic paralysis of the accommodation, as oculists often see it after slight, sometimes not even recognized, angina diphtheritica. Yet, the father's statements gave no explanation. Finally, with hesitation, the father related that about 8 weeks previously the child had had an inflammation with purulent discharge from the genitals and had been treated by a physician.

An answer to a letter the colleague stated that he had seen the child but once and had found discharge and swelling of the labia, as we see it with oxyuris. "He had not thought of diphtheria, since there were no general symptoms." No cases of diphtheria were reported from the neighborhood.

In spite of this I do not doubt for a moment that this was one of those rare cases of vulvovaginitis diphtheritica, for there were absolutely no symptoms of angina diphtheritica and, then, the appearance of the first signs of paralysis in the eyes came on 6 weeks after the suspicious vulvovaginitis, as is usual with post-diphtheritic paralyses. This paralysis disappeared with tonic treatment after a few weeks.

The case is worthy of a record on account of the rare occurrence of diphtheritic vulvovaginitis and the naturally even more rare paralysis of the accommodation after diphtheritis vulvæ. Maybe the general practitioners can by bacteriological examination now and then recognize such an "oxyuris vulvitis" as true diphtheria and prevent further infection by prompt isolation. The oculist will, perhaps, find in rare instances an ætiologically unexplained case of paralysis of the accommodation cleared up when thinking of the ætiology in this case.

*Klin. Mtsbl. f. Augenhlk., LIII, p. 427.

ABSTRACTS FROM MEDICAL LITERATURE.

By W. F. Hardy, M.D.,
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SOME OCULAR MANIFESTATIONS OF AURAL DISTURBANCES AND THEIR INTERPRETATION.

J. A. Hagemann (*Med. Record*, January 16, 1915) contributes an article on the above subject. He states that ocular nystagmus resulting from aural pathology or experiment has been an enigma. It is undisputed that ocular nystagmus may be induced by cranial conditions other than aural. Rotation and caloric tests establish or disprove labyrinthine involvement, but the raison d' être remains abstruse. The nerves and their branches involved in nystagmus are analyzed in regard to their origin relations and distribution. A connection between the cortical centers controling optic muscular movements and the medullar and allied optical centers has not been fully demonstrated. From experimental and pathological observations theoretical association centers have been invoked whose apparent location is in the large ganglionic cells situated about the corpora quadrigemina. From these cells a tract arises which descends toward the medulla and by reason of its relationship to the nuclei of the several nerves which supply the eye muscles, may be regarded as a reflex center. On this hypothesis we can interpret the significant ocular manifestations of auricular irritation. The impression propelled from the semi-circular canals is borne along the course of the auditory nerve. At or near its source the conjectural association centers convert the impressions into motor impulses, which are projected along the several nerves supplying the ocular muscles. Not arising from conscious cortical direction, it would be expected that the resultant muscular movements would be different from those under the guidance of conscious cerebration. These graphic involuntary manifestations are, however, not erratic. The familiar ocular nystagmus is a true reflex. A relation between the middle ear and the eye may be established through the trigeminus. In acute purulent otitis media severe pain may be felt in the eye accompanied by conjunctivitis. This is a transmission, not a reflex, as the impulse passes from one branch to another of the same nerve by a circuitous route, and the resultant paroxysm is really a distal manifestation of the enkindling

excitation. The least prevalent way in which correlation between ear trouble and ocular manifestations may occur is by direct injury of a nerve that supplies one or more ocular muscles. The trochlear, motor oculi and abducens nerves and the Gasserean ganglion lie over the apical end of the temporal bone. The abducens is particularly liable to injury here because it courses in a fibrous or bony gutter or duct upon the outer side of the inferior petrosal sinus to the cavernous sinus, and at this point hugs the bone for perhaps 1 cm. Occasionally a temporal bone will contain a chain of pneumatic cells extending from the vicinity of the middle ear to the apex. It is conceivable therefore that an infective process may traverse this channel, perforate the surface of the bone and invade the nerve fibers lying in close apposition thereto. Injury or destruction of the 6th nerve would then result in paralysis of the externus. Injury of the Gasserian ganglion could produce conjunctivitis, corneal trouble and other grave signs separate from the eye. Paralysis of the trochlear nerve has been reported. Pupillary reactions resulting from ear diseases are not well understood. Inequality of pupils, especially in children, in the absence of apparent other cause, should suggest possible aural trouble.

THE EXTRACTION OF THE CATARACTOUS LENS IN ITS CAPSULE AS PRACTICED IN THE COLTEA HOSPITAL IN BUCHAREST, ROUMANIA.

Howard F. Hansell (*Penn. Med. Jour.*, December, 1914) describes the operation of Prof. Stanculeanu as practiced by him for the relief of cataract. Hansell states that we all agree that the ideal operation is the extraction of the lens in its capsule. The patient escapes the dangers of unexpelled cortex with the accompanying iritis, iridocyclitis, sometimes glaucoma, and fortunately rarely purulent infection. Also the patient is saved months or years of semi-blindness while waiting for the lens to harden. The idea that the cataractous lens of a patient over sixty is hard and its cortex easily freed from its capsule is erroneous. The extraction of the lens in its capsule finds, however, its greatest use in immature cataracts, though the obviating of a secondary operation on capsular remains is of virtually equal importance. Stanculeanu's operation is a follows: On the evening before operation a one per cent. solution of homatropine is dropped in the eye. The incision in the limbus

cornea is somewhat larger than is usually made. With a sickle shaped forceps, the size of the Graefe iris forceps, the anterior chamber is entered. The forceps are introduced closed. When in position the forceps is opened and the largest possible fold of capsule is grasped. By wide horizontal and upward and downward movements the zonula is ruptured. The grasp on the capsule is released, the forceps withdrawn, and the lens expressed with two spatulas. The iris is replaced and eserin instilled. This original procedure was modified by Stanculeanu himself in 1912 by omitting the homatropine and substituting iridectomy. The forceps was also modified to lessen the danger of tearing the capsule. The operation is not suitable for hypermature cataract, that with a small nucleus and milky cortex; in which cases it is almost impossible to pick up a fold of capsule because the branches of the forceps glide over it.

When the capsule tears, which means failure of this special method, the operation is completed in the usual way. Hansell draws attention to the following salient points regarding the Stanculeanu operation: 1. Thorough anæsthesia with 4 per cent. cocaine, instilled six times at four minute intervals, the first and last combined with adrenalin 1:2000. 2. The making of a conjunctival sliding flap. The method of making the flap is described in detail. A suture is inserted on either side, the knot loosely tied and the loops pushed out of the way. The advantages claimed for the flap are, preservation of intact vitreus, rapid closing of the corneal wound, prevention of entrance into the anterior chamber of foreign or septic material, and a safeguard from accidents during convalescence. 3. The use of a double hook for elevating the upper lid, the lower lid being depressed by the first finger of the assistant's free hand. 4. The incision comprises one-half of the corneal circumference and should be made in the corneal rather than the scleral limbus. Healing is as prompt as when made in the scleral limbus and the tendency to sepsis is no greater. 5. A small piece of iris is excised in nearly every case, the exceptions being when the pupil is widely dilated. 6. The removal of the lens in its capsule as described above. Occasionally the manipulations cause the lens capsule to rupture either while the lens is lying in its fossa or while being expressed. 7. The dislocation of the lens consists of four distinct movements. Two lateral forcible enough to bring the lateral periphery into the area of the dilated pupil, downward and finally upwards and forwards. Expression is

performed with a spoon below and a spoon or spatula on the sclera behind the cut. The first pressure is directly backwards to make the superior periphery of the lens present at the section; the spoon is now shifted to the lower limbus and the lens gently expelled. 8. The toilet presents nothing unusual. 9. The final step is to draw the conjunctiva by means of the threads over the upper third of the cornea and to knot them.

According to Hansell, two comprehensive conclusions seem justifiable. First, Stanculeanu's operation is to be recommended only in uncomplicated senile cataracts both mature and immature. Second, it is not to be adapted to the extraction of hypermature cataracts.

THE OCULAR MANIFESTATIONS IN INFANTILE SCURVY.

Sydney Stephenson (*Ophthalmoscope*, March, 1915) comments upon the paucity of reference to this condition in ophthalmological text-books. The commonest signs are (a) protrusion of the eyeball, and (b) hæmorrhage beneath the skin of the eyelids or neighboring parts. These signs may occur separately or together. The proptosis varies from slight to marked, but rarely leads to exposure of the cornea. Likewise the effusion of blood may vary from but a stain of the eyelid to a large protrusion of the eyelid by blood. Stephenson states that these cutaneous or subcutaneous hæmorrhages are due usually to an effusion of blood between the periorbita, on the one hand, and the bones of the orbit on the other, which accounts for the frequent coexistence with proptosis. Barlow pointed out that the painful bone manifestations of infantile scurvy were due to sub-periosteal effusion of blood. Proptosis and ecchymosis may occur with great suddenness, often after a fit of crying. Subconjunctival hæmorrhages may occur. Flame-shaped retinal hæmorrhages and blood in the anterior chamber have been noted. Typical cases are readily diagnosticated. A difficulty might arise, as Stephenson suggests, when the ocular antedates all other signs of scorbutus. It is well then to remember that a black or protruding eye during the first dentition, not due to injury, is more likely to be caused by scurvy than by anything else. A case report is given where the ocular antedated the other signs. It should be stated, however, that while the effused blood rapidly subsided under the influence of an anti-

scorbutic diet, and that later blood occurred in the stools and urine, yet on the other hand the classic signs of infantile scurvy, as revealed by the post-mortem, were conspicuous by their absence.

DOUBLE PAPILLO-ŒDEMA—OPTIC NEURITIS.

Robt. Sattler (*Lancet Clinic,* April 3, 1915) takes up the subject of double papillo-œdema. It is still unsettled whether or not excessive intracranial pressure is only a common or an invariable cause of double papillo-œdema. The pathogenesis remains obscure. Little advance has been made in modern times towards a rational explanation. It is the dominant symptom of a typical syndrome of brain and cerebellar tumors. Sattler urges the early search for and discovery of this ocular symptom in order that surgical treatment may be early instituted; that life and sight may be saved or blindness deferred. He states that the terms papillo-œdema, bilateral optic neuritis, or choked optic discs represent an earlier or later stage, greater or less degree of consecutive pathological progression of a distant and more often undiscoverable location, of a primary intracranial new growth. This phænomenon is met with in from 80 to 90 per cent. of all cases of brain and cerebellar neoplasms. It is frequently discovered accidentally and may exist without impairment of vision. Often it marks the very beginning of an intraocular tumor. It is accompanied by localizing headaches and causeless vomiting, which trinity of symptoms should always arouse attention even in the absence of other clinical symptoms. It is assumed that persistent intracranial hypertension explains double optic neuritis, yet our conceptions of a normal intracranial tension are not on a sound scientific footing. Abnormal and persistent increase or decrease of intracranial pressure is largely a matter of brain dynamics. The factors concerned are an unyielding bony framework, elastic contents, blood and lymph streams, which together with other regional safeguards maintain a more or less normal pressure equilibrium consistent with protection, nutrition and function. Most mechanical theories assume that optic neuritis owes its origin to an increased perverted and displaced volume of cerebro-spinal fluid, which forces its way into the dural and arachnoidal sheaths of the optic nerves. A convexity forwards of the lamina cribrosa is made the dividing line between papillo-œdema and optic neuritis. When bowed in towards the interior of the eye it is assumed that an active

exudative inflammation is present. If this is not present it is regarded as a simple œdema. it is assumed for some focal lesions whether cystic or metastatic, and the more solid growths, that they begin their activity as irritative or degenerative lesions. Sattler makes the further pertinent statement, "It is furthermore assumed that double papillo-œdema, for the reason of its early and often unknown presence or without impairment of vision, is a consequence of the primary concealed focal gliosis wherever present, but through unknown intricate anatomic contact with the visual path or primary optic centers, or many regions adjacent or correlated therewith, or from the latter encroaching through anatomic physiologic contact upon the former, induces a similar and consecutive process, and as an infiltration œdema travels to the lower optic tracts and optic nerves. For anatomical reasons only, one-half or less of each optic nerve is first affected, but by continuity of tissue œdematous infiltration encroaches and conceals the entire nerve head."

The fundamental question is, how to speedily obtain relief. High tension and tumor growth are both destructive, with the difference that one precedes the other in harmful consequences, and that once damage is done even partial or transitory adjustment with a semblance of function may be no longer possible.

A CASE OF SPONTANEOUS RECOVERY FROM DETACHMENT OF THE RETINA.

Herbert L. Eason (*Lancet,* January 2, 1915) records another spontaneous recovery from retinal detachment. These happy results are unfortunately all too few and a persistence of the reattachment is viewed in most quarters with some misgivings. Eason's case was in a woman of 45 years, who suffered sudden loss of vision which the ophthalmoscope proved to be retinal detachment in the left eye. Right eye highly myopic. Circumstances prevented prolonged treatment—rest in bed, pilocarpin and mercurialization, so the patient returned home. Two weeks later, Eason was informed, vision had returned: O.S., which with her glass, —11.00 S, =6/12, reattachment being later confirmed by Eason himself. Field of vision complete, and with the exception of a posterior staphyloma and a rather thin choroid of high myopia, the interior of the eye was normal in every respect. A satisfactory explanation of the sudden loss of vision in detachment and its sudden return on reattachment is not at hand. Eason states that the loss of vision can hardly be due to

any failure of vascular or lymphatic circulation, else the retina would degenerate rapidly and detachment and recovery after an interval would be impossible. The retina strips away leaving the layer of pigment epithelium attached to the choroid, and it seems probable that without the pigment epithelium and possibly the visual purple the other layers of the retina cannot perceive light. Eason thinks that the extremely defective vision of pronounced albinos suggests that the pigment layer of the retina is essential to vision, and that in detachment of the retina it is the separation of the rods and cones from the pigment epithelium that causes the blindness over the area of the detachment.

GLAUCOMA AND THE BLOOD PRESSURE.

Alexander McRae (*Ophthalmoscope,* April, 1915) carried out some experiments and observations in the endeavor to ascertain if there existed any real relation between glaucoma and increased blood pressure. The work of previous observers was reviewed. Hill's theory concerning glaucoma finds favor with McRae. It is to the effect that there is a local toxæmia, bacterial or otherwise, producing chemical changes in the colloids of the eye which cause them to absorb water. This theory is said to meet every factor in the case and needs invoke no help from the blood pressure. The gist of McRae's paper is contained in a paragraph at the end of the article. He says, "These experiments, considered along with the other points brought forward here, suggest that an altogether exaggerated importance has been given to the blood pressure as a factor in maintaining or increasing eye tension. That it does help to maintain the eye tension no one will deny. But the eye tension may be affected in many ways which have no connection with and no effect on blood pressure; and conversely, the blood pressure may alter greatly without any corresponding effect on eye tension being observable. That blood pressure is of any importance at all in the causation of glaucoma is very doubtful. It is at least likely that the solution of the long standing problem of the ætiology of that disease may prove to be a chemical one with the well-known phænomenon of osmosis as its basis."

McRae's paper had to do with primary glaucoma, acute, subacute and chronic. Secondary glaucoma was excluded from all consideration.